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Prognostic Relevance of Subjective Theories of Illness on the Clinical and Psychological Parameters in Irritable Bowel Syndrome Patients – A Longitudinal Study

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1. Introduction

Irritable bowel syndrome (IBS) is a highly prevalent functional gastrointestinal disorder that is characterised by abdominal pain associated with altered stool frequency and/or consistency. The criteria for IBS were last specified by the Rome Committee in 2006 (Drossmann et al., 2006):

Chronic or recurrent abdominal pain or symptoms of not less than three months duration during the last 6 months and associated with not less than 2 of the following 3 criteria: <ul style="list-style-type: none">• improvement in the symptoms following defecation• alteration in stool frequency and/or• alteration in stool consistency
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Table 1. Rome III criteria for the diagnosis of IBS, adapted from Drossman et al., 2006

While the exact cause of IBS is still not known, multiple factors, such as visceral hypersensitivity (alterations in gastrointestinal sensitivity), motility disturbances, psychosocial factors and immune-mediated factors, are thought to contribute to the symptom complex of IBS (Mathew & Bhatia, 2009). These changes seem to be triggered by stress (Mayer et al., 2001). However, there is no uniform, scientifically-based model of the etiology of IBS that patients could use for orientation.

Patients with IBS have been repeatedly described as lacking health-related quality of life compared to healthy controls (Dancey et al., 2002). The majority of patients with IBS symptoms do not consult a healthcare professional. One of the main reasons that patients decline to consult a doctor is not the severity of symptoms but rather the fear of a serious illness (Whitehead et al., 2002) or other psychological or somatic comorbidities (Riedl et al., 2008).

The course of chronic diseases puts high demands on the coping behaviours of patients. To cope with a chronic disease, patients develop explanations, convictions and expectations concerning the origin of the disease that can be subsumed as subjective theories of illness (Faller, 1993). Based on their research into subjective causes, patients retrospectively ascribe a meaning to their illnesses by attempting to understand why they occurred in the first place, whether they could be influenced in the future and how relapses could be prevented. This subjective theorisation contributes to patients’ coping mechanisms (Hampson et al., 1990). Because of these subjective theories of illness, the patient is enabled to make predictions and decisions concerning therapeutic options or the handling of their medical conditions (Faller, 1993). Furthermore, there is evidence that subjective theories of illness affect compliance and cooperation in the relationship between the doctor and the patient (Leventhal et al., 1980) and the restoration of fitness for work (Lacroix et al., 1991, Schiaffino et al., 1998). Subjective causal assumptions are especially important for the sufferers of diseases for which a uniform, evidence-based etiological model providing patients with orientation has not been developed, as is the case for IBS. Subjective causal assumptions and their effects on clinical parameters in IBS and on disease perception are poorly understood and have been insufficiently evaluated. This study is based on an adaptation (Figure 1) of the Common Sense Model of Illness Representation by Leventhal et al. (1980).

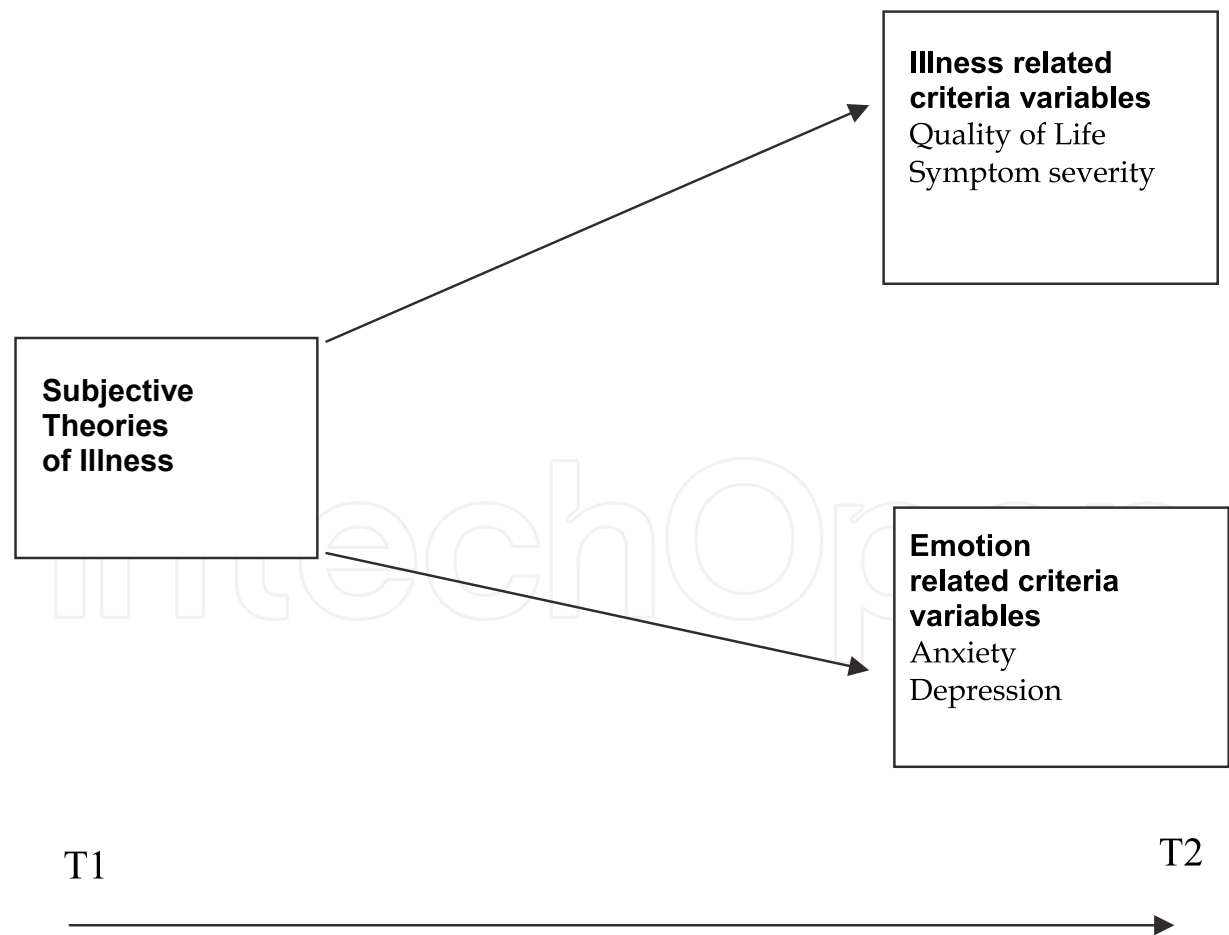


Fig. 1. Adaptation of the Common Sense Model of Illness Representation by Leventhal et al. (1980); Hagger & Orbell (2003)

This model describes the link between subjective theories of illness, coping behaviour and quality of life and is considered the most influential theoretical model regarding the regulatory processes of patients dealing with diseases (Leventhal et al., 1980). Leventhal et al. stated that patients develop ideas about their illnesses within five representational dimensions: identity; causal attributions; expectations of duration; expectations of consequences; and perceived control and curability. The identity of an illness is characterised by its symptoms and the diagnosis itself. The search for the causes of a disease (causal attributions) represents a fundamental part of subjective theories of illness. In addition to this retrospective cognition, these subjective theories of illness also contain prospective assumptions, including expectations of disease duration and its consequences. Leventhal et al. (1980) also described anticipated control as a dimension of subjective theories of illness. In the present study, we focused on the investigation of causal attributions.

Evidence for the validity of the Common Sense Model of Illness Representation (Leventhal et al., 1980) has been found in various cross-sectional studies (Hagger & Orbell, 2003; Rutter & Rutter, 2002). However, few studies have validated this model on the longitudinal axis (Hagger & Orbell, 2003; Kaptein et al., 2003), and the only longitudinal study investigating the influence of subjective theories of illness in IBS patients showed that theories remained stable over one year (Rutter & Rutter, 2007). “Psychological causal assumptions” and “severity of expected consequences” proved to be predictors for anxiety and depression. However, subjective theories of illness were not significantly predictive for the “quality of life” parameter (Rutter & Rutter, 2007). Because they did not examine the possible consequences of subjective theories on clinical symptoms in this study, no conclusion can be drawn concerning somatic outcome.

We recently reported a correlation between subjective theories of illness, symptom severity and quality of life. Due to this study’s cross-sectional study design, we were not able to test for causality at this time (Riedl et al., 2009).

Therefore, we designed the following study to examine whether patients’ causal attributions would have prognostic relevance with regard to symptoms and patient quality of life over the course of an illness.

Based on the Common Sense Model, this study evaluated whether subjective theories of illness in IBS patients could affect clinical outcome parameters, such as symptom severity, quality of life, anxiety and depression, after one year. Furthermore, we evaluated whether subjective theories of illness change over time in IBS patients.

2. Methods

2.1 Measurements

2.1.1 Subjective theories of illness

To encompass a wide range of causal assumptions in addition to the causal assumptions questionnaire (SKT) by Faller (Faller & Walitzer, 2001), we included a second Cause Questionnaire that was developed by Fliege (Fliege et al., 2003) and was originally intended for chronic inflammatory bowel disease (IBD) patients; it was subsequently modified for IBS (Riedl et al., 2009). This latter questionnaire includes the items “dysfunctional stress regulation” and “fatalism”, which are two elements that are not covered by the SKT.

2.1.2 Causal assumptions questionnaire (SKT)

The SKT (Faller & Walitzer, 2001) includes 16 items that are rated on a 5-point response scale. Validated in 197 patients attending a psychotherapist clinic, this questionnaire displays good psychometric characteristics. The items are assigned to four scales by factor analysis. These scales are as follows:

Scale I, Intrapsychic causes, with four items (Could your disorder result from (1) internal conflicts, (2) internal anxiety, (3) poor coping with problems and/or (4) lack of self-confidence?);

Scale II, Social causes, with three items (stress through worries about family and partnership setting, lack of understanding through other people and conflicts with other people);

Scale III, Interpersonal causes, with six items (unhealthy lifestyle, difficulties in professional life, common life stress, environmental stress, financial problems and social circumstances); and

Scale IV, Somatic causes, with three items (somatic disease, poor circulation and weather).

The parameters for internal consistency, expressed as Cronbach's α , are good for scales I and III (scale I: T1 = 0.84, T2 = 0.93; scale III: T1 = 0.7, T2 = 0.68) and satisfactory for scales II and IV (scale II: T1 = 0.76, T2 = 0.57; scale IV: T1 = 0.56, T2 = 0.57).

2.1.3 Cause questionnaire for chronic IBD patients

For our purposes, the original questionnaire developed by Fliege et al. (2003) to assess causal assumptions in IBS patients required a change of instructions. The term chronic IBD was replaced with IBS, but the original wording of the items was retained. The questionnaire includes 16 items that are assigned to five scales (dysfunctional stress regulation, interactionality, lifestyle, physiology and fatalism) with 5-point response formats. For the present study, only two scales were included ("dysfunctional stress regulation" and "fatalism") to enrich the SKT, which lacks these items.

The scale "fatalism" only comprises two items: higher power and destiny/fate (Spearman's r : T1 = 0.56, T2 = 0.55).

The scale "dysfunctional stress regulation" is based on four items: wrong stress coping, high workload, inability to relax and disposition to be overly sensitive to negative situations (Cronbach's α : T1 = 0.76, T2 = 0.78).

2.1.4 Outcome

2.1.5 Beck Depression Inventory (BDI)

The BDI (Hautzinger, 1991) is a self-rating scale for the assessment of depressive symptoms; it contains 21 items (Cronbach's α : T1 = 0.86, T2 = 0.72).

2.1.6 Hospital Anxiety and Depression Scale (HADS)

The HADS (Herrmann, 1997), which is a 14-item scale, was specifically developed for use by individuals with somatic diseases. It measures patients degrees of anxiety and depression based on their self-ratings. For the present study, only the anxiety scale was used (Cronbach's α : T1 = 0.76, T2 = 0.78).

2.1.7 Health-related quality of life (SF-12)

The SF-12 (Bullinger, 1996) is a brief version of the SF-36, which measures two components of quality of life: mental well being and physical condition (Cronbach's α : T1 = 0.67 and 0.64, T2 = 0.66 and 0.71).

2.1.8 Questionnaire for gastrointestinal symptoms

The somatic parameters were assessed with the aid of a questionnaire that was specifically developed for and routinely employed by the Outpatient Clinic for Gastrointestinal Motility Disorders and Functional Gastrointestinal Diseases at Charité University Medical Center. Subjects were asked to check the frequency and severity of 13 symptoms that are typical of irritable bowel syndrome (pain, pressure, cramps in the upper and lower abdomen, the feeling of being bloated, the unpleasant passing of gas, diarrhoea, constipation, alternating diarrhoea and constipation, the feeling that the bowels have not been completely evacuated and loud or bothersome bowel sounds [borborygmi]).

The total scores were calculated as the sum of the products of frequencies ("How frequently have you suffered from your symptoms", rated on a scale from 0 = never to 4 = constantly) and severities ("How bad were your symptoms", rated on a scale from 0 = no symptoms to 5 = very severe) of the monitored symptoms (Cronbach's α : T1 = 0.88, T2 = 0.75).

3. Participants and study design

Patients received questionnaires on hand-held computers during their waits at the Outpatient Clinic for Gastrointestinal Motility Disorders and Functional Gastrointestinal Diseases at Charité-University Medical Center, Campus Virchow (Berlin, Germany). Patients were included in the study based on the Rome III criteria following a medical examination (all subtypes were included using the Bristol stool scale). The criteria for exclusion were as follows: age <18 years, lack of cooperation and the indication of another disease explaining the symptoms (such as chronic inflammatory bowel disease (IBD), lactose or fructose intolerance, malignant disease or the abuse of alcohol other drugs).

After the criteria for inclusion or exclusion had been checked, 88 patients were able to be included in the study at T1. Patient recruitment at T1 took place from November 2006 to March 2007. Questionnaires were mailed to these 88 patients after one year for follow-up evaluations (T2, November 2007 to March 2008). Of these patients, 49 subjects returned the questionnaire, and 3 had moved to unknown addresses. Of the 49 data sets received, 44 were completed and included in the follow-up study.

The sample comprised 30 females (68%) and 14 males (32%). The mean age was 46 years (SD = 15.23 years, range = 19-72 years).

4. Statistical analysis

All analyses were carried out with the statistics program SPSS, version 14.0. The descriptive parameters were calculated for each scale, and the normal distribution was assessed by the Kolmogorov-Smirnov test.

Intercorrelation was calculated by the Pearson correlation coefficient, and predictors for clinical and psychological outcome criteria of causal assumptions were tested by multiple stepwise linear regression analyses. All scales covering the subjective theories of illness at T1 that significantly correlated with the criteria variables at T2 (see Table 2) were included in the regression analyses. Furthermore, the base level (T1) of criteria variables was enclosed as an autoregressor in all regression analyses. Other predicting effects that exceeded the autoregressor can be considered to be robust.

5. Results

5.1 Characteristics of causal assumptions at T1 and T2

The SKT was dominated by the “intrapsychic causes scale” (M = 2.50, SD = 0.18 at T1 and M = 2.55, SD = 0.17 at T2) and the “social causes scale” (M = 1.89, SD = 0.11 at T1 and M = 2.02, SD = 0.97 at T2), followed by “somatic causal attributions” (M = 1.78, SD = 0.11 at T1 and M = 2.08, SD = 0.12 at T2) and “interpersonal causal attributions” (M = 1.80, SD = 0.13 at T1 and M = 2.02, SD = 0.13 at T2). In the causal assumptions questionnaire (SKT), the highest mean corresponded to the scale “dysfunctional stress regulation” (M = 1.97, SD = 0.15 at T1 and M = 2.07, SD = 0.13 at T2). The “fatalism scale” produced the lowest mean of all scales (M = 0.51, SD = 0.13 at T1 and M = 0.55, SD = 0.12 at T2).

5.2 Stability of causal assumptions over the course of one year

To assess the stability of causal assumptions over the course of one year, mean comparisons were performed using a t-test. Except for the scale “somatic causes” (p = 0.01), there were no significant changes supporting consistency in the attribution of causal assumptions. The score of “somatic causes” increased after one year.

5.3 Correlations and regression analyses

Table 2 depicts the correlations of causal assumptions at T1 and outcome at T2.

	<i>Physical QoL¹</i> <i>at T2</i>	<i>Mental QoL¹</i> <i>at T2</i>	<i>Anxiety</i> <i>at T2</i>	<i>Depression</i> <i>at T2</i>	<i>Symptom</i> <i>severity at T2</i>
<i>Causal assumptions at T1</i>					
Dysfunctional Stressregulation	.31*	-.41**	.65**	.26	.36*
Fatalism	-.23	-.36*	.44*	.53*	.25
Intrapsychic causes	.21	-.39**	.61**	.44**	.34*
Interpersonal causes	.07	-.44**	.58**	.51	.27
Social causes	.03	-.29	.44**	.19	.47**
Somatic causes	-.37*	.02	.12	.01	.21

¹QoL = Quality of life
Note * p < 0.05, ** p < 0.01

Table 2. Correlations between causal assumptions at T1 and outcome variables at T2.

No differences between causal assumptions with regard to age, gender or IBS-subgroups were found. Thus, these socio-demographic variables were not considered for later analysis. To examine the overall relationship between the predictor variables and the outcome variables, all correlating predictors were entered stepwise in a linear regression analysis. Table 3 shows the last step of each regression. β (beta coefficient) is the standardised regression coefficient of the last step of each regression. It shows, which regressor (in this

Outcome Symptom Severity					
Predictors	β	R^2 Step 1	ΔR^2 Step 2		
Social causes	0.33*	0.22 (p < 0.01)			
Symptom intensity at T1	0.43**		0.17 (p < 0.01)		
Outcome Depression					
Predictors	β	R^2 Step 1	ΔR^2 Step 2	ΔR^2 Step 3	
Fatalism	0.05	0.31 (p < 0.001)			
Interpersonal causes	0.15		0.08 (p < 0.05)		
Value depression at T1	0.74***			0.35 (p < 0.001)	
Outcome Anxiety					
Predictors	β	R^2 Step 1	ΔR^2 Step 2	ΔR^2 Step 3	
Dysfunctional stress regulation	0.25*	0.42 (p < 0.001)			
Interpersonal causes	0.25*		0.12 (p < 0.001)		
Anxiety at T1	0.46**			0.11 (p < 0.001)	
Outcome Mental Quality of Life					
Predictors	β	R^2 Step 1	ΔR^2 Step 2	ΔR^2 Step 3	
Interpersonal causes	0.25	0.21 (p < 0.01)			
Dysfunctional stress regulation	-0.09		0.08 (p < 0.05)		
Psychological quality of life at T1	0.71***			0.27 (p < 0.001)	
Outcome Physical Quality of Life					
Predictors	β	R^2 Step 1	ΔR^2 Step 2	ΔR^2 Step 3	ΔR^2 Step 4
Somatic causes	-0.13	0.13 (p < 0.05)			
Dysfunctional stress regulation	0.28*		0.11 (p < 0.05)		
Fatalism	-0.35*			0.08 (p < 0.05)	
Physical quality of life at T1	0.49**				0.18 (p < 0.001)

β = standardised coefficient; Regression value of the last step
*** < 0.001, **p = 0.01; * p < 0.05.

Table 3. Regressions of causal assumptions and autoregressor (T1) of all outcome variables (T2).

case subjective causal assumptions) makes the maximum contribution to the prognosis of the dependent variable (in this case outcome variables).

R^2 , the coefficient of determination, is a measure of the portion of the variance explained, ΔR^2 stands for the changes in the amount of the variance explained effected by the subjective causal assumptions entered in the last step.

5.3.1 Causal assumptions and IBS symptom severity

In addition to the autoregressor, symptom severity was only predicated by the scale “social causes” ($\beta = 0.33$, $p < 0.05$). Social causal assumptions accounted for 22% of the variance in symptom severity at T2.

5.3.2 Causal assumptions and depression

The first step showed the scale “fatalism” to be a significant predictor for depression. It explained 31% of the recorded variance (step 1: $\beta = 0.55$; $p < 0.001$). Another 8% of this variance was explained in the second step by the scale “interpersonal reasons” (step 2: $\beta = 0.34$; $p < 0.05$). In tests, the influence of causal assumptions was not significant beyond the influence of the base level of depression at T1. Thus, the base level for depression proved to be the only significant predictor of depression at T2 ($\beta = 0.74$; $p < 0.001$) (35% of variance explained).

5.3.3 Causal assumptions and anxiety

Causal assumptions explained 54% of the variance of “anxiety” at T2 (Table 3). Furthermore, the scales “dysfunctional stress regulation” and “interpersonal causes” were equally prognostic for “anxiety”.

5.3.4 Causal assumptions and mental quality of life

Causal assumptions explained 29% of the variance, and the base level explained another 27% of the variance. No significant effects of the causal assumptions “interpersonal cause” (step 1: $\beta = -0.46$, $p < 0.01$; step 2: $\beta = -0.35$, $p < 0.05$) and “dysfunctional stress regulation” (step 2: $\beta = -0.30$, $p < 0.05$) were found after checking for the base level of mental quality of life at T1. Thus, no significant effect of causal assumptions on mental quality of life was found after considering the autoregressor.

5.3.5 Causal assumptions and physical quality of life

The scales “dysfunctional stress regulation” and “fatalism” significantly predicted patients’ physical quality of life at T2 (19% of variance explained).

6. Discussion

In this study, we longitudinally assessed subjective theories of illness in IBS and their prognostic relevance based on the Common Sense Model of Illness. Two questions were asked: 1.) do subjective theories of illness in IBS change over time, and 2.) can they predict clinical and psychological outcomes?

All causal items had relatively small means and standard deviations, indicating that patients likely give little significance to single causes. This finding may represent a study limitation. It might be possible that the questionnaires that were used did not properly reflect causal

assumptions. “Intrapsychic factors”, “social factors” and “dysfunctional stress regulation” received the strongest agreement. In IBS patients, causal assumptions that were based on stress and intrapsychic factors are particularly relevant because they reflect scientific aetiology models that show connections between coping experience and symptom reinforcement.

We assessed beliefs at two time points. Except for the scale “somatic causes”, the results of the comparisons of the means of causal assumptions at T1 and T2 were not significant. Therefore, our results indicate a consistency of causal assumptions over time and support known data regarding IBS (Rutter & Rutter, 2007). Leventhal et al. (1980) argued that patients’ illness representations are constantly being updated as new illness experiences and knowledge are acquired. This argument cannot be supported by our findings, except for those in the “somatic causes” scale, possibly due to the fact that over time, IBS patients increase their knowledge about scientific, physical explanations for their symptoms. Similar to Rutter & Rutter (2007), we predominantly found no change in individuals’ illness representations across the two measured time points. This finding supports the hypothesis that IBS is a chronic disease and distinguishes it from Leventhal’s model, which was designed largely around acute illnesses. The stability of illness cognitions in IBS patients over time is an important clinical finding that, if replicated in further, larger studies, will provide strong indicators for psychological interventions with these patients.

While Rutter and Rutter (2007) used longitudinal data but only predicted psychological outcomes, we focused this study on the prediction of variables of clinical outcomes such as symptom severity, allowing us to demonstrate for the first time that subjective theories of illness have prognostic relevance in IBS. Through these results, we show a strong predictive value of subjective theories of illness based on symptom severity and psychological factors (anxiety).

Via the direct regression path, subjective causal assumptions explained 22% of the variance in symptom severity, in which particularly social causes significantly predicted symptom severity after one year. Patients who strongly attributed their illnesses to social causes (stress through worries in family and partnership setting, lack of understanding through other people and conflicts with other people) reported elevated symptom severity after one year.

Furthermore, subjective causal assumptions explained 54% of the variance in anxiety after one year. Patients who attributed the cause of their disease to dysfunctional stress regulation (coping with stress incorrectly, high workload, inability to relax or disposition to be overly sensitive to negative situations) or interpersonal causes (unhealthy lifestyle, difficulties in professional life, common life stress, environmental stress, financial problems or social circumstances) showed higher values for anxiety after one year.

Subjective theories of illness proved to be predictive with regard to physical quality of life but not predictive of mental quality of life. Patients who attributed the cause of their illnesses to fatalism (higher power and destiny/fate) reported an impaired physical quality of life, while causal attribution to dysfunctional stress regulation resulted in an improvement of physical quality of life after one year.

Subjective theories of illness were not predictive for depression during the course of the study. This finding could be due to the fact that depression is considered to be more of a chronic comorbidity in IBS patients and is thus a stable variable that is weakly influenced by subjective theories of illness. However, in the first step of the regression analysis, 31% of the

variance for depression was explained by fatalist causal assumptions, and only after adjusting for the base level did this predictive value become insignificant, suggesting that fatalism and depression may be confounded in these responses.

Our results confirm the presumption of the Common Sense Model of Illness because they show that subjective theories of illness have direct and fundamental prognostic value for clinical and psychological factors in IBS. The longitudinal prognostic relations of subjective theories of illness to symptom severity, anxiety and physical quality of life are still preserved when the base level of criteria variables is adjusted for statistical controls. Thus, the effect of the predictors can be considered robust.

Our findings point to the relevance of subjective causal assumptions in IBS by revealing several partly differential connections between single causal dimensions and outcome variables. Some of the study weaknesses include a relatively small sample size and a significant loss to follow-up. The small sample size is limiting given the number of variables that could be included in the regressions.

7. Conclusion

This study of the treatment of IBS patients is significant with regard to the information gain that occurs in subjective theories of illness and to the relevance of these theories for symptoms of anxiety and depression and for quality of life and symptom severity.

Social causal attributions that relate to interpersonal and domestic conflicts are prognostic for perceived increased symptom severity. It should be noted that subjective theories of illness point to a worsened disease course in IBS patients, which should be sufficient to include maladaptive causal assumptions in the focus of (psycho-) therapeutic treatment.

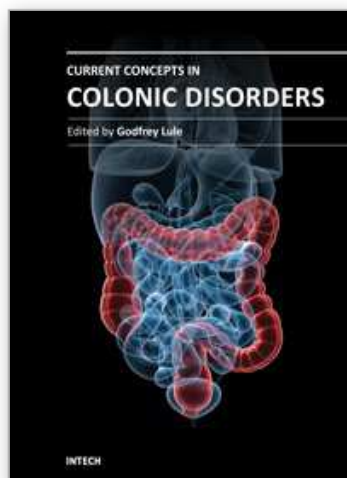
Dysfunctional stress regulation (coping with stress incorrectly, high workload, inability to relax or disposition to be overly sensitive to negative situations) and interpersonal causes (unhealthy lifestyle, difficulties in professional life, common life stress, environmental stress, financial problems or social circumstances) have prognostic value for the increased occurrence of anxieties. This causal relation could be the product of a chronic distress burden caused by real social problems and therefore might be considered an indirect prognostic factor for the development of anxiety in IBS. IBS patients have a lifetime prevalence (up to 90%) of developing a psychiatric comorbidity such as an anxiety disorder (Lydiard et al., 1993). Thus, it becomes evident that the knowledge of prognostic factors, such as subjective theories of illness, is valuable for preventive and therapeutic care. To counteract the development of anxious or depressive symptoms in the course of IBS, adverse subjective theories of illness should be recognised as early as possible and adapted if necessary.

The medical histories of patients with IBS should include an assessment of subjective theories of illness. It seems especially important that, for the clinical course of the disease, physicians inquire about and treat adverse prognostic factors in the social environment (stress through worries in family and partnership setting, lack of understanding through other people and conflicts with other people) as early as possible. The knowledge of the content and significance of the concepts that patients develop to explain their disease will enable healthcare professionals to better understand treatment approaches and patient expectations and improve doctor-patient relationships in the treatment of irritable bowel syndrome.

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The 21st Century has seen a resurgence of research of the gastrointestinal tract, especially since it was established that it plays a central role as an immune system organ and consequentially has a huge impact on causation, impact and transmission of most human ailments. New diseases such as the Acquired Immunodeficiency Syndrome, hepatitis and tumours of the gastrointestinal tract have emerged and they are currently subjects of intensive research and topics of scientific papers published worldwide. Old diseases like diarrhea have become extremely complex to diagnose with new and old pathogens, drugs, tumours and malabsorptive disorders accounting for the confusion. This book has set out algorithms on how to approach such conditions in a systematic way both to reach a diagnosis and to make patient management cheaper and more efficient. "Current Concepts in Colonic Disorders" attempts to put all the new information into proper perspective with emphasis on aetiopathogenesis and providing rational approach to management of various old and new diseases. As the book editor, I have found this first edition extremely interesting and easy to understand. Comments on how to improve the content and manner of presentation for future editions are extremely welcome.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

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